

MERCURY, VACCINES, AND AUTISM, REVISITED

Baker's recent article¹ presented fascinating insights and perspectives on the intertwined stories of mercury, vaccines, and autism. As a researcher in mercury with some involvement in the autism issue (as a participant in expert committees for the National Institutes of Health, the National Research Council, and the Environmental Protection Agency [EPA] as well as an invited reviewer of a proposed clinical trial of mercury chelation in autism), I would like to offer some additions to an excellent article.

First, it is not entirely correct to suggest that there is no medical knowledge of the potential hazards associated with thimerosal apart from a convergence with the history of knowledge about methyl mercury. Baker omits the separate (but convergent) history of toxicities associated with thimerosal in topical medicines, such as contact lens solution, eye drops, and other products. The literature on this history (first reviewed in 1981²) prompted restrictions on the use of thimerosal in these products by the Food and Drug Administration (FDA) in 1998.

Second, most methyl mercury exposure occurs because of the consumption of fish that has been exposed to environmental biomethylation. Methyl mercury has not been used in paints or pesticides; the organomercurials that have been used in these products are methoxy ethyl mercury chloride and phenyl mercury compounds.³

Third, although there is experimental evidence that ethylmercury behaves differently from methyl mercury in terms of toxicokinetics,⁴ it appears to have qualitatively similar effects on the nervous and immune systems.^{5,6}

Finally, an additional force for convergence not mentioned by Baker is the coincidence of the increasing number of early childhood vaccinations with the increasing knowledge of low-level organomercury toxicity.

Although Baker's article is informative, I read history somewhat differently. I believe it illustrates an additional lesson not noted in the article: much trouble could have been avoided if the FDA had made a prudent decision early in the controversy to reduce infant exposures to mercury compounds by removing

thimerosal from medications. Such a decision should have been made at least by 1997, when the EPA issued its report to Congress on mercury.⁷

This path would have emulated the voluntary cessation of lead soldering in food cans, which the FDA encouraged and the food industry undertook in the early 1970s without a prolonged debate on whether this specific use was associated with specific neurotoxic outcomes in children. To quote my former mentor J. Julian Chisolm, "one should not shy from introducing interim measures" even if they are partial.⁸ Mercury, like lead, is a chronic and accumulative toxin, and reductions in any source have a public health benefit. No discussion of specific associations with autism would have been necessary, and much heartache could have been avoided. The increased suspicion of the public toward the biomedical profession, the drug industry, and the regulatory community could have been avoided as well.

Moreover, the intensity of the advocacy response, particularly by parents of children with autism, should be seen in the context of the lack of attention to preventable risk factors for autism, which is clearly not a genetically determined disease although genetic susceptibility may play an important role in modulating response to acquired risks. In this sense, mercury may be seen as symbolic of the importance of environmental risk factors (defined broadly and not just chemically) as well as of the lack of a research agenda at the national level for autism despite its status as a major neurodevelopmental disorder of children. ■

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